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Race and sex differences in associations of vegetables, fruits, and carotenoids with lung cancer risk in New Jersey (United States)

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We used data from a case-control study conducted in New Jersey between 1980 and 1983 to evaluate race and sex differences in associations of vegetable, fruit, and carotenoid consumption with lung cancer. Cases included 736 White males, 860 White females, 269 Black males, and 86 Black females with incident, histologically confirmed, primary cancer of the trachea, bronchus, or lung. Controls were identified through drivers' license and Health Care Financing Administration files and included 548 White males, 473 White females, 170 Black males, and 47 Black females. Usual intakes of vegetables (predominantly yellow/green) and fruit (predominantly yellow/orange) as well as other food sources of carotenoids were ascertained by a food frequency questionnaire. White females showed significant inverse associations of lung cancer with vegetables, fruit, and carotenoids. White males showed nonsignificant inverse associations with vegetables and carotenoids, and Black females just with vegetables. No inverse associations were found for Black males. Vegetable consumption was associated with risk of all histologic types of lung cancer, but the pattern of increasing risk with decreasing intake was limited to smokers. We infer that consumption of yellow/green vegetables and carotenoids may confer protection from lung cancer to White male and White female smokers. Further studies are needed to clarify the effect in Blacks.

Key words: Blacks, carotenoids, diet, fruit, lung neoplasms, sex, United States, vegetables, Whites.

Introduction

Vegetable[1-8] and fruit[1-3,8-10] consumption has been related to a reduced risk of lung cancer in numerous epidemiologic studies. Carotenoids—potent antioxidants found primarily in dark green and yellow vegetables, and yellow and orange fruits—also have been reported to protect against lung cancer.[1,2,6,10-18] Inverse associations with vegetables, fruits, and carotenoids have been reported more frequently and more consistently in males than in females,[5,6,11,14,15] and the one study[2] that investigated Black/White differences was inconclusive.

During the 1980s, the incidence rate of lung cancer in men was twice that in women.[19] Although comparable rates were seen for White and Black women, the rate in

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Black men was 50 percent higher than in White men,[19] and not all of this disparity could be explained by dissimilarities in smoking patterns.[20] We, therefore, used data from a large case-control study conducted in New Jersey (United States) between 1980 and 1983 to evaluate whether discrepancies in lung-cancer incidence rates could be accounted for, in part, by differences in associations of vegetables, fruit, and carotenoids with lung cancer in males and females and in Whites and Blacks. Ziegler *et al*[21,22] previously reported results from this study for White males.

Materials and methods

Study population

Case-control data were collected in three phases in New Jersey between 1980 and 1983. Cases for phase one included all male residents of six, high lung-cancer-mortality areas with incident, histologically confirmed, primary cancer of the trachea, bronchus, or lung (International Classification of Diseases[23] code 162) diagnosed between 1 September 1980 and 31 October 1981. Methods have been described in detail by Ziegler *et al*[22] and Schoenberg *et al.*[24] Briefly, cases were ascertained through local hospitals, the New Jersey cancer registry, and mortality files. For cases who were interviewed directly, controls were selected randomly from New Jersey drivers' license files and frequency matched to cases by age (five-year intervals), race, and area of residence. For cases for whom surrogates were interviewed because of death or incapacitation, controls were selected from New Jersey mortality files. To increase the number of Black males, phase two extended the study to cover all incident lung cancers occurring among Black males between 1 August 1982 and 30 November 1983. Controls were selected using the same procedures as phase one except that a telephone-screening questionnaire was used to ascertain race, a variable which was not included on drivers' license files. Phase three identified incident lung cancer cases among females residing statewide between 1 August 1982 and 30 September 1983. Similar methods to the other two phases were used except that for directly interviewed cases aged 65 or older, controls were selected randomly from Health Care Financing Administration (HCFA) files.[25]

Interviews were obtained directly for 57 percent of cases, with the remaining interviews completed by surrogates including 48 percent spouses, 36 percent children, 10 percent siblings, and six percent other relatives. Smoking and dietary patterns did not vary by respondent type, except that fruit consumption was reported more frequently by living cases than for deceased cases. As reported by others,[26] however, a much higher smoking prevalence was reported for deceased controls identified through mortality files than was reported by living controls identified through drivers' license and HCFA files, even when the coded cause of death was not clearly related to smoking. Because controls identified through mortality files have different health-risk profiles from the general population, as exemplified by their higher smoking prevalence, they do not represent the population from which cases arose and their inclusion could bias risk estimates.[27] We, therefore, used only controls selected from drivers' license files and HCFA files in this analysis.

Data collection

Usual diet, smoking, smoking by other household members (passive smoking), occupation, residential and medical histories, and demographic characteristics were ascertained by in-person interviews. Diet was assessed using a food frequency questionnaire which ascertained usual frequency of consumption, three to five years earlier, of selected foods. The questionnaire used in phase one of the study emphasized the food sources of carotenoids. Although the questionnaire was expanded to include additional vegetables and fruits for the later phases, only the foods included in both questionnaires were used in analyses. These were the vegetables (predominantly yellow/green) and fruits (predominantly yellow/orange) listed in the Appendix, dairy products, and mixed dishes containing these items.

For fruits and vegetables that were eaten seasonally, in-season and out-of-season frequencies of consumption and season length were used to estimate mean numbers of servings per month during an entire year. Frequencies of individual vegetables and fruits were summed to obtain vegetable- and fruit-group frequencies, respectively. A carotenoid index was calculated by summing the carotenoid content of usual servings of food items after weighting by the frequency of consumption. Carotenoid values were derived from the US Department of Agriculture Nutrient Database for Food Consumption Surveys.[28] Since the food frequency questionnaire did not ascertain information on portion sizes, median portion sizes reported by individuals 50 to 75 years of age, in 24-hour dietary recalls, collected as part of the Second National Health and Nutrition Examination Survey, were used to estimate the size of a typical serving.[29] The same values of carotenoid content per usual serving were used for all race/sex groups.

Subjects could not always recall if a food item was eaten, or, if eaten, how often it was eaten. Seventeen (one percent) White males, 33 (two percent) White

females, 21 (five percent) Black males, and four (three percent) Black females had nonquantifiable responses for five or more food items and were excluded from analyses. Median values for all races and sexes combined were substituted for missing data for individuals with fewer than five nonquantifiable responses. Less than one percent of frequencies of consumption of food items was imputed to any race/sex group. Eight subjects who were missing data on smoking or education also were excluded from analyses.

Analysis

We used multiple logistic regression to estimate odds ratios (OR) and 95 percent confidence intervals. All models were adjusted for race and/or sex, age, education, employment in high-risk occupations,[30] passive smoking, and personal smoking. Individuals were categorized as: nonsmokers; pipe/cigar smokers only; former cigarette smokers, if they quit more than a year prior to diagnosis of lung cancer for cases or interview for controls; current cigarette smokers, if they smoked at the time of diagnosis or interview or had quit within a year; light smokers, if they smoked an average of less than 20 cigarettes a day; and heavy smokers, if they smoked an average of 20 or more cigarettes a day. For males, adjustment also was made for area of residence (see map in reference 24). Because Black males were included in both the first and second study phases, an indicator variable was included in models with Black males to adjust for differences between phases.

Statistical significance of trends was assessed by substituting median values for tertiles and performing a chi-square test.[31] Effect modification was evaluated using median values of tertiles to calculate cross-products terms separately with race and with sex and testing the significance of these terms in logistic models. All analyses were performed using SAS statistical software.[32]

Results

Interviews were completed for 73 percent of cases but only 62 percent of controls. After excluding subjects with missing data, 70 percent of cases and 61 percent of controls could be included in analyses. Table 1 shows the numbers and percentages of cases and controls included for each race-sex group.

Inclusion of all cases, but only living controls, resulted in surrogate interviews being used just for cases. Interviews were completed by surrogates for 43 percent of White males, 46 percent of White females, 35 percent of Black males, and 40 percent of Black females.

Characteristics of cases and controls by race and sex are shown in Table 2. A greater proportion of lung cancers were squamous cell carcinomas in males compared with females, while adenocarcinomas (including bronchioalveolar) were more common in females than in males. Significant case-control differences in smoking histories were observed among all race/sex groups.

Lower, middle, and upper tertiles of intake of yellow/green vegetables, yellow/orange fruits, and carotenoids were defined by the frequency distribution in all controls combined. Distributions of controls in tertiles for each race/sex group are shown in Table 3. White males and females were approximately equally divided among tertiles for all dietary categories, except for an excess of White females in the upper tertile for vegetables. Black male controls were more likely to be in the lower tertiles for vegetables and for fruits; but because Black male controls had a high frequency of intake of some carotenoid-rich vegetables, such as pumpkin/yams and greens, they were not more likely to be in the lower tertile for carotenoids.

Lung cancer ORs by tertile of consumption for yellow/green vegetables, yellow/orange fruits, and carotenoids are shown in Table 4. Consumption of vegetables and carotenoids was associated inversely with lung cancer risk in analyses with and without surrogate interviews, with the strongest, most consistent, association for vegetables. The remainder of analyses also were performed with and without surrogate interviews for cases, but because findings did not differ substantially, only results including surrogates are reported.

In analyses by race/sex group, significant inverse trends in lung cancer risk among White females were observed for yellow/green vegetables, yellow/orange fruits, and carotenoids (Table 5). White males in the lower tertile had nonsignificant increased risks for vegetable and carotenoid intake. The small number of Black females also had nonsignificant elevated risks in the lower tertiles for vegetables. Among Black males, however, lower intake was associated with a reduced risk of lung cancer for each dietary category. Effect modification by race was statistically significant for fruit and carotenoids and effect modification by sex was significant for fruit.

Table 1. Number and percent of cases and controls included in dietary analyses by race and sex

	Cases	Controls
White males	736 (69%)	548 (63%)
White females	860 (74%)	473 (63%)
Black males	269 (63%)	170 (50%)
Black females	86 (72%)	47 (61%)

Table 2. Percent distribution of subjects by demographic characteristics, smoking, and histology for race/sex groups

	White males		White females		Black males		Black females	
	Cases (No. = 736)	Controls (No. = 548)	Cases (No. = 860)	Controls (No. = 473)	Cases (No. = 269)	Controls (No. = 170)	Cases (No. = 86)	Controls (No. = 47)
Age								
< 60 years	32	31	30	34	41	48	59	51
60-69 years	41	37	37	39	35	32	24	30
70 + years	28	31	32	27	24	21	16	19
Education								
< 12 years	64	44[b]	45	34[b]	75	60[a]	59	45
Occupation								
Ever high-risk	41	32[a]	1	1	53	46	5	4
Smoking status								
Nonsmoker	2	17[b]	12	51[b]	1	21[b]	8	43[b]
Pipe/cigar only	3	10	0	0	1	11	0	0
Former light	6	17	9	16	8	25	10	17
Former heavy	26	25	8	7	10	8	3	4
Current light	11	10	26	17	35	25	45	32
Current heavy	52	21	45	8	43	11	33	4
Histology								
Squamous	49	—	27	—	47	—	22	—
Adenocarcinoma	16	—	29	—	14	—	36	—
Small or oat cell	15	—	23	—	16	—	14	—
Other or not specified	20	—	21	—	23	—	28	—

[a] $P < 0.01$.
[b] $P < 0.001$.

Table 3. Percent distribution of controls by tertiles for vegetable, fruit, and carotenoid intake

	All (No. = 1,238)	White males (No. = 548)	White females (No. = 473)	Black males (No. = 170)	Black females (No. = 47)
Vegetables (servings/mo.)					
66 +	33	30	40	25	32
42-65	33	36	32	29	34
≤ 41	33	34	28	46	34
Fruits (servings/mo.)					
37 +	33	35	36	22	23
19-36	33	32	36	29	36
≤ 18	33	32	28	49	40
Carotenoids (RE/mo.)[a]					
16,735 +	33	28	37	38	38
9,684-16,734	33	37	32	28	23
≤ 9,683	33	34	31	34	38

[a] RE = retinol equivalents.

Since vegetable consumption showed the strongest inverse association with lung cancer and food values for carotenoids are imprecise,[33] we performed analysis by histologic type using yellow/green vegetables as the exposure. Because of small numbers, analyses of histologic type-specific differences in associations could not be performed simultaneously by race and sex. ORs for all subjects combined and by race and by sex separately are presented in Table 6. Among all subjects combined and Whites alone, risk decreased with increasing yellow/green vegetable consumption for all histologic types of lung cancer and trends were significant for squamous, adenocarcinoma, and small and oat cell carcinoma. In Blacks, however, only a nonsignificant inverse gradient in risk for squamous cell carcinoma was observed. In males, nonsignificant trends of increasing risk with decreasing intake were apparent for squamous and other cell types, but in females, lower vegetable consumption was associated with an increased risk of lung cancer for all histologic types.

Table 4. Odds ratios[a] (95% confidence intervals) for lung cancer by dietary intake including and excluding surrogate interviews for cases

	Including surrogates (1,951 cases, 1,238 controls)	Excluding surrogates (1,111 cases, 1,238 controls)
Vegetables (servings/mo.)		
66+	1.00	1.00
42-65	1.25	1.18
	(1.10-1.54)	(0.93-1.50)
≤41	1.37[d]	1.37[d]
	(1.12-1.69)	(1.08-1.73)
Fruits (servings/mo.)		
37+	1.00	1.00
19-36	1.15	1.13
	(0.93-1.41)	(0.89-1.42)
≤18	1.15	1.00
	(0.94-1.41)	(0.80-1.27)
Carotenoids (RE/mo.)[b]		
16,735+	1.00	1.00
9,684-16,734	1.08	1.04
	(0.87-1.33)	(0.82-1.33)
≤9,683	1.27[c]	1.20
	(1.03-1.56)	(0.95-1.51)

[a] Odds ratios from logistic models including age, race, sex, education, occupation, residence, smoking, passive smoking, study phase.
[b] RE = retinol equivalents.
[c] P-trend < 0.05.
[d] P-trend < 0.01.

We investigated the joint effect of smoking and consumption of yellow/green vegetables on risk of lung cancer using nonsmokers and former light smokers in the upper tertile of consumption as the reference (Table 7). Nonsmokers and former light smokers were collapsed into a single category because there were few cases who were nonsmokers and no dietary associations were observed in either category when analyses were performed on all race/sex groups combined. Whites who were former heavy, current light, and current heavy smokers and who had low vegetable intakes were at an increased risk of lung cancer, but Blacks with low consumption were not at an increased risk in any smoking stratum. Males and females who had low vegetable intakes were at an increased risk of lung cancer if they were current light or current heavy smokers, and females were at an increased risk if they were former heavy smokers.

Discussion

Our findings support a protective effect of yellow/green vegetables and carotenoids for lung cancer in White males and White females. Vegetables also were weakly inversely associated with lung cancer risk in the small number of Black females. In Black males, however, none of the dietary measures was related to risk.

Table 5. Odds ratios[a] (95% confidence intervals) for lung cancer by dietary intake for race/sex groups

	White males (736 cases, 548 controls)	White females (860 cases, 473 controls)	Black males (269 cases, 170 controls)	Black females (86 cases, 47 controls)
Vegetables (servings/mo.)				
66+	1.00	1.00	1.00	1.00
42-65	1.05	1.56	0.89	1.72
	(0.76-1.45)	(1.13-2.17)	(0.45-1.74)	(0.57-5.17)
≤41	1.25	1.70[d]	0.91	1.50
	(0.91-1.71)	(1.22-2.36)	(0.49-1.70)	(0.51-4.40)
Fruits (servings/mo.)				
37+	1.00	1.00	1.00	1.00
19-36	1.25	1.31	0.59	0.99
	(0.92-1.70)	(0.94-1.82)	(0.30-1.15)	(0.33-3.02)
≤18	1.05	1.77[d]	0.49[c]	0.77
	(0.77-1.42)	(1.26-2.47)	(0.27-0.90)	(0.26-2.28)
Carotenoids (RE/mo.)[b]				
16,735+	1.00	1.00	1.00	1.00
9,684-16,734	1.01	1.43	0.73	0.77
	(0.73-1.39)	(1.02-2.00)	(0.40-1.34)	(0.25-2.38)
≤9,683	1.20	1.80[e]	0.79	0.67
	(0.87-1.65)	(1.30-2.50)	(0.45-1.40)	(0.25-1.80)

[a] Relative odds from logistic models including age, education, occupation, residence (males), smoking, passive smoking, study phase (Black males).
[b] RE = retinol equivalents.
[c] P-trend < 0.05.
[d] P-trend < 0.01.
[e] P-trend < 0.001.

Table 6. Odds ratios[a] (95% confidence intervals) for lung cancer by histology and vegetable intake for all subjects and for race and sex groups

Vegetable servings per month	Squamous	Adenocarcinoma	Small or oat cell	Other or not specified
All				
66 +	1.00	1.00	1.00	1.00
42-65	1.28	1.26	1.11	1.28
	(0.97-1.68)	(0.92-1.72)	(0.77-1.60)	(0.93-1.77)
≤41	1.53[c]	1.38[b]	1.60[c]	1.30
	(1.17-2.01)	(1.01-1.88)	(1.13-2.27)	(0.94-1.79)
Whites				
66 +	1.00	1.00	1.00	1.00
42-65	1.33	1.26	1.07	1.25
	(0.99-1.80)	(0.90-1.77)	(0.72-1.60)	(0.88-1.78)
≤41	1.61[c]	1.54[b]	1.76[c]	1.36
	(1.20-2.16)	(1.10-2.15)	(1.20-2.59)	(0.95-1.94)
Blacks				
66 +	1.00	1.00	1.00	1.00
42-65	1.12	1.34	1.30	1.46
	(0.54-2.32)	(0.55-3.30)	(0.47-3.60)	(0.62-3.44)
≤41	1.27	0.83	0.96	1.21
	(0.66-2.43)	(0.35-1.95)	(0.38-2.42)	(0.54-2.73)
Males				
66 +	1.00	1.00	1.00	1.00
42-65	1.16	0.73	0.88	1.17
	(0.82-1.65)	(0.44-1.19)	(0.52-1.46)	(0.74-1.85)
≤41	1.28	0.98	1.15	1.36
	(0.91-1.79)	(0.61-1.56)	(0.71-1.86)	(0.87-2.12)
Females				
66 +	1.00	1.00	1.00	1.00
42-65	1.47	1.84	1.34	1.46
	(0.92-2.36)	(1.23-2.77)	(0.79-2.28)	(0.93-2.31)
≤41	2.15[d]	1.84[c]	2.24[c]	1.23
	(1.37-3.39)	(1.21-2.79)	(1.32-3.77)	(0.76-1.98)

[a] Relative odds from logistic models including race and/or sex, age, education, occupation, residence (males), smoking, passive smoking, study phase (Blacks and males).
[b] P-trend < 0.05.
[c] P-trend < 0.01.
[d] P-trend < 0.001.

These findings could not be explained by differences in histologic type of lung cancer or by smoking patterns.

Only one other study[2] has investigated Black-White differences in lung cancer risk associated with carotenoid intake and, in that study, a slight inverse gradient in risk of squamous and small cell cancer was evident in both Blacks and Whites. The racial differences that we observed in associations of diet with lung cancer could be due to genetic variation. Alternatively, methodologic shortcomings of our study could explain these differences.

Interindividual differences in susceptibility to lung cancer, at least in part, are inherited,[34] and the Black-White differences in the role of diet that we observed, could be due to genetic variation. Several epidemiologic studies have reported an increased risk of lung cancer among family members of lung cancer cases.[35-37] Different frequencies of *ras* oncogene alleles among cases[38] and evidence for Mendelian inheritance of a rare autosomal gene[39] also have been reported. A difference in metabolism of carcinogens has been suggested as a mechanism underlying genetic variation in lung cancer susceptibility.[34]

The lack of an apparent association between diet and lung cancer in Blacks in our study also could be due to the smaller number of Blacks and/or bias. Only 50 percent of potential Black male controls could be included in analyses compared with more than 60 percent in other race/sex groups, suggesting the possibility of selection bias. The phrasing of particular questions and the omission of some foods from the food frequency questionnaire also may have led to misclassification of Blacks. For instance, collards, turnip greens, and mustard greens are among the primary source of carotenoids for Blacks,[40] yet they were grouped into a single item labelled 'greens' on our questionnaire. The term

Table 7. Odds ratios[a] (95% confidence intervals) for lung cancer by smoking status and vegetable intake for all subjects and for race and sex groups

Vegetable servings per month	Smoking status				
	Nonsmokers or former light	Pipe or cigar only	Former heavy	Current light	Current heavy
All (1,951 cases, 1,238 controls)					
66 +	1.0	0.7 (0.2-2.3)	4.5 (2.9-6.9)	5.1 (3.3-7.8)	14.3 (9.5-21.5)
42-65	1.3 (0.9-1.9)	1.5 (0.7-3.5)	6.0 (3.9-9.1)	5.4 (3.6-7.9)	17.1 (11.4-25.6)
≤ 41	1.0 (0.7-1.5)	1.1 (0.5-2.4)	6.6 (4.3-10.4)	8.4 (5.7-12.5)	22.6 (15.4-33.2)
Whites (1,596 cases, 1,021 controls)					
66 +	1.0	0.7 (0.2-2.7)	4.8 (3.0-7.7)	4.6 (2.8-7.4)	14.9 (9.6-23.1)
42-65	1.5 (1.0-2.2)	2.6 (1.1-6.4)	5.9 (3.8-9.3)	5.6 (3.6-8.7)	16.0 (10.4-24.8)
≤ 41	1.1 (0.7-1.6)	1.3 (0.6-3.0)	7.2 (4.5-11.6)	9.1 (5.7-14.6)	24.5 (16.0-37.3)
Blacks (355 cases, 217 controls)					
66 +	1.0	—	2.6 (0.7-9.7)	7.0 (2.6-18.9)	14.2 (4.3-47.5)
42-65	0.8 (0.3-2.0)	—	10.0 (2.5-39.8)	4.7 (1.9-11.7)	36.6 (10.5-128.5)
≤ 41	0.8 (0.3-2.1)	—	3.7 (1.0-14.1)	6.8 (2.9-15.9)	17.0 (6.4-45.4)
Males (1,005 cases, 718 controls)					
66 +	1.0	0.6 (0.2-1.9)	4.4 (2.4-8.1)	3.8 (1.9-7.6)	11.2 (6.2-20.4)
42-65	0.9 (0.5-1.8)	1.1 (0.5-2.8)	4.4 (2.5-7.8)	4.4 (2.3-8.3)	10.9 (6.2-19.5)
≤ 41	0.7 (0.4-1.4)	0.8 (0.4-1.9)	4.9 (2.7-9.0)	6.8 (3.7-12.5)	14.4 (8.3-24.9)
Females (946 cases, 520 controls)					
66 +	1.0	—	2.3 (1.1-4.8)	5.9 (3.5-10.2)	15.6 (8.6-28.4)
42-65	1.6 (1.0-2.4)	—	8.0 (3.4-19.0)	5.7 (3.5-9.4)	30.8 (15.3-61.9)
≤ 41	1.2 (0.8-1.9)	—	8.0 (3.4-18.8)	9.1 (5.4-15.4)	43.5 (21.8-87.0)

[a] Relative odds from logistic models including race and/or sex, age, education, occupation, residence (males), passive smoking, study phase (Blacks and males). Because only 4 Black male cases just smoked pipes or cigars, we included pipe and cigar smoking in the logistic model for Blacks as a single dichotomous variable and did not calculate odds ratios by tertile of vegetable intake.

'greens' may not be precise enough for people to accurately quantify intake,[41] and thus we may have underestimated vegetable and carotenoid consumption among Blacks. Additionally, oranges and orange juice, which are more important sources of carotenoids for Blacks than Whites,[40] were not included in our questionnaire.

In our series, lung cancer risk was associated inversely with yellow/green vegetable and carotenoid consumption for White males and White females and with yellow/orange fruit consumption for White females. Six studies have reported associations of vegetables, fruit, or carotenoids with lung cancer separately for both males and females. Le Marchand *et al*[6] observed trends in males and females similar to ours, but in four other studies[2,5,14,15] associations were limited to males. In a study by Byers *et al*,[11] carotenoids from fruits and vegetables were associated inversely with lung cancer in White males overall and in White females who were nonsmokers. Differences in reported findings by sex could be due to the smaller number of female subjects included in most studies or differences in smoking histories of males and females.

The pattern of increasing risk of lung cancer with decreasing consumption of yellow/green vegetables that we observed was limited to smokers. Carotenoids can be potent antioxidants and may react with a carcinogen in cigarette smoke, thereby protecting against

lung cancer in cigarette smokers only. Such an effect would be consistent with the lower blood β-carotene levels noted in smokers relative to nonsmokers with comparable carotenoid intakes.[42,43]

Associations between diet and lung cancer have been reported to differ by smoking status in numerous epidemiologic studies but not in a consistent manner. Associations have been reported to be strongest in long-term smokers,[17] heavy smokers,[44] light smokers,[2] current smokers,[7] former smokers,[16] and nonsmokers.[10,11] Definitions of dietary intake and of smoking categories vary among studies and many studies do not disentangle heavy smokers from long-term and current smokers which could contribute to these discrepancies.

Most investigators have reported inverse trends with dietary intake of vegetables, fruits, and carotenoids for multiple histologic types of lung cancer.[4,6,7,11] In our series, also, low yellow/green vegetable consumption was associated with an increased risk of all histologic types of lung cancer in Whites. In addition to acting possibly as antioxidants, carotenoids may protect against lung cancer by other mechanisms, including maintaining normal differentiation after conversion to retinoids at the cellular level and stimulating the immune system.[45] The lack of specificity of dietary associations for a particular histologic type of lung cancer may be related to the multiple actions of carotenoids with widespread effects.

In conclusion, risk of lung cancer in our series increased with decreasing intakes of yellow/green vegetables in White males and White females. Inverse associations were observed between vegetable intake and all histologic types of lung cancer but were restricted to smokers. We infer that yellow/green vegetable intake protects against lung cancer in White smokers. Additional studies are needed to clarify the effect in Blacks.

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Appendix. Foods included in vegetable and fruit categories

Vegetables	Sweet red peppers
Asparagus	Winter squash
Broccoli	Yams/sweet potatoes
Carrots	
Corn	**Fruits**
Green beans	Cantaloupe
Green cabbage	Peaches/nectarines
Green peas	Apricots
Greens	Grapefruit juice
Head lettuce	Prunes
Leaf lettuce	Tomatoes (fresh)[a]
Summer squash	Tomato juice[a]
Sweet green peppers	Watermelon

[a] Botanically a fruit.